Stevens–Johnson Syndrome Associated with Beza® Brate

Sir,

Beza® Brate is a fibric acid derivative, widely used for patients with hyperlipidemia, and is considered to be a safe medication (1). We report here the first case of Stevens–Johnson syndrome associated with beza® Brate treatment.

CASE REPORT

A 42-year-old female with slight hyperlipidemia was treated with beza® Brate in the hospital of her general practitioner. On the 13th day of treatment she noticed fever, joint pain and exanthema on her extremities. She stopped taking beza® Brate and referred herself to the same hospital. She was treated with an oral steroid and antibiotics and the symptoms disappeared within a week. As there was no strong indication not to take beza® Brate, she restarted taking the drug and 5 days later developed an erythematous eruption on the whole of the body, fever, joint pain and edema of the face. Examination revealed multiple generalized erythematous macules, target lesions and erosions on her trunk and extremities. We also found pigmentation resulting from previous skin lesions. She had shallow erosions on her lips and hard palate, and redness of her conjunctiva. At admission to our hospital, laboratory tests revealed a low hemoglobin level (9.6 g/dl). The leukocyte count was 14,000/mm³, with 82% polymorphs, 14% lymphocytes and 4% monocytes. Blood biochemistry and urine and stool examinations were normal. Serological tests for mycoplasma and herpes viruses were negative. Chest X-ray and electrocardiogram were normal. A biopsy specimen revealed hydropic degeneration of basal cells and numerous scattered necrotic keratinocytes with eosinophilic cytoplasm (colloid bodies) in the epidermis. A dense superficial perivascular mononuclear cell infiltrate was found around superficial blood vessels. In some areas, hydropic degeneration caused subepidermal separation and all keratinocytes appeared necrotic.

We made a diagnosis of Stevens–Johnson syndrome caused by beza® Brate. The patient was treated with oral prednisolone (30 mg/day) and azelastine hydrochloride (2 mg/day). After 3 days her symptoms were alleviated and the erythema and erosion began to heal. Oral prednisolone was tapered and the patient was discharged 10 days later. A skin patch test and a drug lymphocyte stimulation test for allergy to beza® Brate were negative.

DISCUSSION

Fever, joint pain and involvement of conjunctival and labial mucous membranes indicated erythema multiforme major, i.e. Stevens–Johnson syndrome. The present case clearly indicated that beza® Brate caused this eruption, although skin patch and drug lymphocyte stimulation tests were negative. However, clofibrate, another fibric acid derivative, has been found to induce erythema multiforme (2) and Stevens–Johnson syndrome (3).

REFERENCES


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